of which are associated with the development of gout. The authors suggest that treatment differences might be caused by physicians being more vigilant when treating women with gout, because women are more likely to have comorbidities.

Original article Harrold LR *et al.* (2006) Sex differences in gout epidemiology: evaluation and treatment. *Ann Rheum Dis* **65:** 1368–1372

Mycophenolate mofetil is a promising treatment for persistent proteinuria in SLE

The optimum treatment for patients with membranous lupus nephritis is uncertain. Several therapeutic regimens have been suggested, including corticosteroids plus cyclophosphamide or chlorambucil; however, these regimens are associated with serious side effects. Mycophenolate mofetil reduces proteinuria in animal models of membranous nephropathy; Borba and colleagues, therefore, assessed the efficacy and tolerability of mycophenolate mofetil treatment of persistent proteinuria in patients with systemic lupus erythematosus (SLE).

In this 18-month pilot study, 20 patients with SLE and persistent isolated severe proteinuria or proteinuric flare (12 of whom had biopsy-proven membranous glomerulonephritis) received open-label mycophenolate mofetil 1.5 g daily for 1 month, escalated to a maximum of 3 g daily or until proteinuria decreased. At enrollment, all patients were taking antimalarial agents and angiotensin-converting-enzyme inhibitors, which were continued throughout the study. Immunosuppressive therapies other than prednisone were discontinued.

Mycophenolate mofetil was well tolerated and associated with few side effects. All patients achieved a partial response (a decrease in proteinuria of ≥50% from baseline) after a mean of 8.2 ± 3.3 months of 2.3 ± 0.5 g mycophenolate mofetil daily. An initial increase of proteinuria was observed in three patients at 4.6 ± 1.5 months; these patients responded to an intravenous dose of methylprednisolone and an increased dose of mycophenolate mofetil. Proteinuria had normalized (<0.3 g per 24 h) in 11 patients at 12.2 ± 3.0 months.

The authors concluded that mycophenolate mofetil could be an effective therapy for patients with SLE who experience persistent proteinuria. Prospective, randomized, controlled trials are warranted.

Original article Borba EF *et al.* (2006) Mycophenolate mofetil is effective in reducing lupus glomerulonephritis proteinuria. *Rheumatol Int* **26:** 1078–1083

BDNF polymorphism is a potential biomarker of cognitive dysfunction in SLE

Impairments in memory, learning and visuospatial skills occur in 20–80% of patients with systemic lupus erythematosus (SLE). Brain-derived neurotrophic factor (BDNF) maintains adult neuronal viability, and animal studies have demonstrated its involvement in short-term and long-term modulation of synaptic plasticity, a crucial process in learning and memory formation. Humans who carry the BDNF Met66 allele of the functional Val66Met polymorphism have impaired hippocampal memory processing; Oroszi and colleagues, therefore, investigated the effect of the BDNF Val66Met polymorphism on cognitive functioning in patients with SLE.

In total, 59 of 60 consecutive SLE patients (53% white, 27% African American, 20% Asian or Hispanic) were assessed. These patients had cognitive function scores in the low-normal to mildly impaired range, although all ethnic groups had a mean of 14.9 years' education. These patients performed most poorly in memory and visuospatial domains. Compared with individuals homozygous for the Val66 allele, those with one or two Met66 alleles showed markedly less cognitive dysfunction in motor and psychomotor domains, which might indicate that the Met66 allele has protective effects. The authors speculate that protection could be achieved by impaired intracellular or extracellular processing of BDNF propeptide, which could alter the balance between (proapoptotic) BDNF propeptide and (antiapoptotic) mature BDNF, and inhibit neuronal apoptosis.

These findings provide preliminary evidence that the BDNF Met66 allele protects against neurocognitive dysfunction in SLE patients. The Val66Met polymorphism could be used to assess SLE patients' risk of developing cognitive dysfunction.

Original article Oroszi G *et al.* (2006) The Met66 allele of the functional Val66Met polymorphism in the brain-derived neurotrophic factor gene confers protection against neurocognitive dysfunction in systemic lupus erythematosus. *Ann Rheum Dis* **65:** 1330–1335